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LETTERS TO THE EDITOR

RE: "DOES TEA AFFECT CARDIOVASCULAR DISEASE? A META-ANALYSIS"

Although there are many commonalities regarding the etiology of vascular diseases, commonalities should not be assumed. This point is illustrated in the paper by Peters et al. (1). These authors performed a meta-analysis of tea consumption in relation to stroke and coronary heart disease, and they found that the study-specific effect estimates for stroke and coronary heart disease were too heterogeneous to be summarized. For stroke, the heterogeneity between risk estimates for tea consumption was suggested to be due to either the geographic region (Australia) or the study design. However, because the only case-control study was also the only study from this geographic region, they were unable to suggest which of these two would be the most likely to explain the heterogeneity. Although these are valid hypotheses, there is a more important and plausible explanation for the heterogeneity observed.

Stroke comprises three distinct subtypes: cerebral infarction, subarachnoid hemorrhage, and intracerebral hemorrhage. These subtypes of stroke are not homogeneous but have differing risk profiles (2–4), incidence rates (5), management (6, 7), and outcomes (8–10). All of the studies of stroke used in the meta-analysis (1), with the exception of the Australian case-control study (3), included all three subtypes of stroke. Given that the most common form of stroke is cerebral infarction, usually comprising 73–86 percent of all strokes (5), it is likely that the risk estimates for tea consumption in these studies were largely influenced by the effect of tea consumption on this particular form of stroke. In contrast, the Australian study was undertaken among patients with fatal and nonfatal intracerebral hemorrhage, a less common form of stroke comprising 8–15 percent of all strokes (5). Because of the differing etiology of this type of stroke compared with cerebral infarction, it is plausible that the effect of tea consumption on these two major types of stroke will be different, thus providing an important explanation for the observed heterogeneity between studies. This interesting disparity is certainly worthy of further investigation.

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THE AUTHORS REPLY

Drs. Thrift and Donnan (1) have pointed out that the heterogeneity among risk estimates for tea consumption and stroke, as described in our meta-analysis (2), might also be explained by differences in subtypes of stroke. To address this valid concern, we divided studies by subtypes of stroke if sufficient information was provided. For only intracerebral hemorrhage did more than one study provide an effect estimate for tea consumption. Whereas one study (3) reported an increased risk of 51 percent for an increment of three cups/day of tea consumption, the other two studies reported decreases of 7 percent (4) and 30 percent (5). The three estimates were not homogeneous ($p = 0.07$). Factors such as study design or geographic region might explain part of this heterogeneity. We agree that it would be helpful for studies of cardiovascular and cerebrovascular events to be more specific and more consistent in their outcome definitions.

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